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Medical Management of Selected Patients With Left Ventricular Free Wall Rupture During Acute Myocardial Infarction

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Objectives. **This study sought to evaluate the effects of prolonged rest and blood pressure control on survival of patients in whom left ventricular free wall rupture (LVFWR) was strongly suspected.**

Background. **Left ventricular free wall rupture in myocardial infarction is often fatal, and only a few patients may undergo operation. However, survival without surgical repair has not yet been evaluated.**

Methods. **Eighty-one consecutive patients with a first transmural acute myocardial infarction in Killip class I or II who presented with acute hypotension due to cardiac tamponade, with electromechanical dissociation (EMD) in 72, were prospectively evaluated. Patients with early recovery were managed with prolonged bed rest and blood pressure control with beta-blockade as tolerated.**

Results. **Forty-seven patients died within 2 h of acute tamponade, and autopsy in 21 showed LVFWR in all. In 15 others, an emergency surgical repair resulted in 2 survivors. The remaining 19 patients, 10 with EMD, had early recovery with dobutamine and colloid solution, and 15 required pericardiocentesis. Shortly thereafter, these 19 patients still showed a paradoxic pulse ≥20 mm Hg, relevant pericardial effusion (24 ± 7 mm [mean ± SD]) and comparable elevation of right and left ventricular filling pressures (15.8 ± 3.9 and 15.9 ± 3.8 mm Hg, respectively). Subsequent management included bed rest (8.2 ± 4.8 days) and control of systolic blood pressure (≤120 mm Hg) with beta-adrenergic blocking agents as tolerated (n = 12). Four patients died, and autopsy in three revealed a rupture that was sealed in two. A sealed rupture was also seen at thoracotomy in 2 other patients who, like the remaining 13, survived for 52.5 ± 35.2 months.**

Conclusions. **Long-term survival of selected patients with prompt hemodynamic recovery after LVFWR is possible without surgical repair. Prolonged bed rest and blood pressure control are likely to contribute favorably to their initial outcome.**

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Acute left ventricular free wall rupture (LVFWR) is increasingly recognized as the second most common cause of mortality in acute myocardial infarction (AMI) (1,2), particularly among patients with a first transmural AMI (2,3). Most patients are identified by the presence of electromechanical dissociation (EMD) (4–8), especially when not preceded by overt heart failure, (8,9) and survival is associated with emergency operation (5,10–14). The subacute form of LVFWR, which presents as acute or progressive hypotension associated with pericardial tamponade but without cardiac arrest, has also been treated surgically, although with better results (10,14,15–18). However, survival remains <50% (10,18,19). In contrast, cardiac rupture may be clinically undetected and lead to pseudoaneurysm (20–23). We hypothesized that some cases of rupture might seal through pericardial adhesions without the formation of a pseudoaneurysm. Therefore, we present this report on 19 patients with a high suspicion of LVFWR who were managed without surgical repair.

Methods

Patients. From March 1986 through May 1995, diagnosis of LVFWR was made in 81 consecutive patients among 1,487 admitted with a first AMI in Killip classes I and II (24). Diagnostic criteria for AMI included the presence of chest pain >30 min unresponsive to sublingual nitroglycerin, ST segment elevation (≥1.0 mm in leads I, II, II, aVL or aVF or ≥2.0 mm in leads V_1 to V_6) and an enzyme level increase at least twice normal values.

Diagnosis of LVFWR. In 72 patients, the clinical diagnosis of LVFWR was based on the development of sudden EMD in the absence of preceding overt heart failure, associated with slowing of heart rate, acutely distended neck veins and cyanosis of the neck and head. The other nine patients did not present with EMD but developed an acute, nonarrhythmic hypotension (systolic blood pressure ≤80 mm Hg). Sixty-two patients with EMD failed to recover from resuscitation maneuvers. Because of the availability of a cardiac surgeon, 15 of them

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Abbreviations and Acronyms

AMI = acute myocardial infarction
EMD = electromechanical dissociation
LVFWR = left ventricular free wall rupture

underwent an emergency thoracotomy that confirmed LVFWR in all (Group A), and suture of the rupture site resulted in 2 long-term survivors. The other 47 patients who failed to recover could not be operated on, and all died within 2 h (Group B). An autopsy study performed in 21 of them confirmed LVFWR in all. The remaining 10 patients and the 9 without EMD recovered from the initial maneuvers. These 19 patients constituted the medically treated group of LVFWR (Group C).

Protocol for medically treated patients. Nineteen patients were treated with intravenous volume (colloid solution preferred) and dobutamine (at 5 to 30 μg/kg body weight per min). The subset presenting with EMD were additionally treated with cardiac massage and mechanical ventilation as needed. If spontaneous pulse was not recovered, pericardiocentesis was attempted. Once the suspicion of LVFWR was entertained, the surgical team was alerted to the possibility of an intervention.

After the acute event, two-dimensional echocardiography was carried out to asses the presence of pericardial effusion, which was measured at the parasternal transversal plane and graded (in mm) as the sum of anterior and posterior effusions. Echograding of right atrial compression was also performed with regard to its presence (+) or absence (0). Right heart catheterization was carried out to monitor right and left ventricular filling pressures and to guide therapeutic measures. Diagnosis of LVFWR was completed by the presence of a paradoxic pulse (≥20 mm Hg) and an increased right atrial pressure (≥10 mm Hg) with a comparable pulmonary capillary wedge pressure (within 5 mm Hg difference). In each instance, echocardiographic evidence of a moderate to severe pericardial effusion was also required (≥10 mm). Patients with right ventricular dilation (diameter >30 mm), associated septal rupture or with the suspicion of right ventricular perforation by a pacing catheter were excluded.

Subsequent management included maintenance of inotropic support as needed, with early attempts at weaning within the ensuing 24 h. This was followed by institution of beta-blockade therapy with oral propranolol starting at 10 mg every 6 h and gradually increasing to 160 mg a day, as tolerated, to keep systolic blood pressure between 100 and 120 mm Hg. Heparin was withdrawn during the first few days, and patients were managed with strict bed rest for at least 5 days and were not allowed to walk for a least 10 days. Routinely, most patients with an uncomplicated first transmural AMI would remain in bed for the first 2 to 4 days and would receive oral beta-blockers starting at days 4 to 8. Two-dimensional echocardiograms were repeated within the first 5 days and serially thereafter, depending on the persistence of pericardial effusion.

Left heart catheterization, including coronary angiography, was performed at least 10 days after LVFWR. Follow-up included clinical surveillance and performance of two-dimensional echocardiography 6 to 12 months later and yearly thereafter, to assess the course of the pericardial effusion and development of pseudoaneurysm. Patients received beta-blocker therapy as tolerated.

Statistics. Statistical comparisons of numerical data were performed by the Student *t* test for paired or unpaired data as appropriate. Two-way chi-square analysis was used to compare categoric variables. Results are reported as mean value ± SD.

Results

Clinical data. Relevant clinical data from the 19 medically treated patients (Group C) are shown in Table 1. Eleven patients (57.9%) presented with transient atrial fibrillation (n = 8) or atrial flutter (n = 3) after diagnosis of LVFWR. In no instance was a pacing catheter introduced before or after the episode of suspected LVFWR. The incidence of anterior AMI was significantly lower than that in patients who did not recover from resuscitation and who did not undergo operation (Group B, $p < 0.02$), whereas the incidence of lateral AMI was significantly higher ($p < 0.01$) (Table 2).

Echocardiographic and hemodynamic data (group C). Mean pericardial effusion was 24.1 ± 6.8 mm, and right atrial compression was present in 16 patients (Table 3). An echodense mass in the pericardial space consistent with a fibrinous cap attached to the infarcted area was observed in 10 patients (Fig. 1 and 2). In one patient, the site of rupture was visualized by echocardiography (Fig. 1), and blood flow to the pericardial sac was confirmed by color Doppler. In another patient, the pericardial effusion was also shown by computerized tomography (Fig. 3). Systolic arterial blood pressure before LVFWR (n = 19) was 133.6 ± 24.1 mm Hg (range 100 to 180). During hypotension or EMD, heart rate decreased in nearly all patients. However, 3 to 4 h later, it increased from 75.9 ± 12.2 beats/min before to 116.3 ± 20.0 beats/min after LVFWR (n = 19, $p < 0.001$), suggesting some degree of persistent tamponade. Right heart catheterization revealed elevated right atrial pressure (15.8 ± 3.9 mm Hg) with comparable pulmonary capillary wedge pressure (15.9 ± 3.8 mm Hg) (Table 3). Central venous pressure measurements available in seven patients shortly before and after diagnosis of LVFWR showed an increase from 4.4 ± 1.8 to 13.7 ± 2.9 mm Hg ($p < 0.0001$). All 19 patients presented a paradoxic pulse >20 mm Hg, and treatment included an intravenous infusion of dobutamine and colloid solutions in all. In addition, cardiac massage was performed in 5 patients, and pericardiocentesis carried out in 15 produced a bloody fluid with a hematocrit close to systemic blood and resulting in a remarkable hemodynamic improvement in 14.

Hospital course. Pericardiocentesis was again performed in three patients on days 2 (Patient 11), 8 (Patient 15) and 9

Table 1. Clinical Data in Medically Treated Patients (group C)

Pt No.	Age (yr)/ Gender	HTN	DM	AMI Site	Days to LVFWR	EMD	Mech Ventil	In-Hosp Survival	F/U (mo)
1	59/M	+	0	Inf	8	0	0	+	120
2	65/M	0	0	Inf	2	0	0	60 d	—
3	65/M	+	0	Lat	4	+	0	+	111
4	54/M	0	0	Inf	1	+	+	+	103
5	63/M	0	0	Inf	3	+	+	+	74
6	59/M	0	0	Lat	4	+	0	+	70
7	75/M	+	0	Lat	1	+	+	9 d	—
8	69/F	0	+	Ant	6	+	+	+	52
9	66/F	0	0	Ant	8	0	0	+	49
10	77/M	+	0	Ant	2	0	0	+	47
11	56/M	0	0	Lat	5	0	0	+	41
12	55/M	+	0	Lat	2	+	0	+	36
13	76/M	+	0	Lat	1	+	0	+	25
14	73/M	0	0	Inf	14	+	+	+	33
15	74/F	+	+	Ant	1	0	+	28 d	—
16	57/M	0	0	Ant	2	0	+	+	12
17	61/M	+	+	Inf	1	0	0	13 d	—
18	51/M	0	0	Ant	1	0	0	+	8
19	73/F	+	+	Lat	1	0	0	+	7

AMI = acute myocardial infarction; Ant = anterior; d = days; DM = diabetes mellitus; EMD = electromechanical dissociation; F = female; F/U = follow-up; HTN = history of arterial hypertension; Inf = inferior; In-Hosp = in hospital; Lat = lateral; LVFWR = left ventricular free wall rupture; M = male; Mech Ventil = mechanical ventilation; Pt = patient; — = not applicable.

(Patient 16) and again on day 10 (Patient 15). Dobutamine infusion was maintained for 3.2 ± 1.9 days, and oral propranolol was started on day 7 ± 4 (range 2 to 14) but was not tolerated by seven patients, mainly because of hypotension. Maintenance dose was 78 ± 26 mg daily. Other agents used were angiotensin-converting enzyme inhibitors in three patients, two of whom were not receiving propranolol, and calcium channel blocking agents in one. Duration of bed rest was 8.2 ± 4.8 days, and ambulation was started at 16.7 ± 5.6 days. Only two patients exhibited a systolic blood pressure ≥140 mm Hg in the sequential measurements during the first 15 days. Four patients died; Patient 2 died of a septic shock on day 60 after LVFWR, and Patient 7 died on day 9 of a progressive respiratory failure due to a pulmonary infection. In both patients, autopsy revealed a sealed LVFWR in a lateral AMI, with extensive pericardial adhesions most prominent at the rupture site. The remaining two patients died of EMD due to a second LVFWR on days 13 (Patient 17) and 28 (Patient 15) despite beta-blocker treatment. However, Patient 15 had undergone pericardiocentesis three times, with a total of 205 ml withdrawn. After extubation 3 days before her death, her tendency to arterial hypertension was difficult to control. Autopsy revealed two areas of LVFWR within an apical infarction, one sealed and the other opened, with endocardial and epicardial thrombi. Patient 17, who presented with a localized posterobasal aneurysm, or false aneurysm, was judged by the surgical team to be at extremely high operative risk given the location of the LVFWR. His blood pressure before death was 150/90 mm Hg, and he died during a postural change.

Two of the 15 survivors (Patients 1 and 18) underwent a thoracotomy, and a sealed LVFWR was confirmed. Patient 1 underwent coronary artery bypass graft surgery on day 42, and Patient 18 underwent a thoracotomy for a pericardial drainage, but because the rupture was sealed, no suture was performed. Nevertheless, to avoid a potential rerupture, Patient 18 was kept sedated and under mechanical ventilation for 7 days. The remaining 13 survivors presented no further complications. Patient 12 is representative of the survivors and his course is illustrated in Figure 4.

Coronary angiographic and left ventriculographic findings. Coronary anatomy was available at 18.9 ± 7.1 days after LVFWR in all but 2 patients (15 by angiography, 2 by postmortem examination). Twelve patients had one-vessel

Table 2. Clinical Data for Groups A, B and C

Group	Age (yr) (mean ± SD)	Female Gender (%)	Days to LVFWR (mean ± SD)	Infarct Site [no. (%) of pts]		
				Ant	Inf	Lat
A (n = 15)	65 ± 7	13	2.2 ± 1.5	7 (47%)	7 (47%)	1 (7%)
B (n = 47)	65 ± 8	32	3.0 ± 3.6	31 (66%)*	13 (28%)	3 (6%)†
C (n = 19)	65 ± 8	21	3.5 ± 3.3	6 (31%)*	6 (31%)	7 (37%)†

*$p < 0.02$. †$p < 0.01$. pts = patients; other abbreviations as in Table 1.

Table 3. Echocardiographic and Hemodynamic Data

Pt No.	Pericardial Effusion (mm)	Right Atrial Compression	Fibrinous Cap	RAP (mm Hg)	PCWP (mm Hg)
1	30	0	0	13	15
2	20	+	+	14	14
3	24	+	0	20	17
4	10	0	0	9	10
5	18	+	0	15	14
6	28	0	+	14	16
7	15	+	+	17	19
8	38	+	0	12	12
9	36	+	0	13	12
10	26	+	0	14	14
11	30	+	+	15	16
12	26	+	0	18	14
13	24	+	+	18	22
14	28	+	0	14	18
15	23	+	+	12	13
16	17	+	+	25	25
17	26	+	+	15	11
18	18	+	+	20	18
19	20	+	+	15	11

PCWP = pulmonary capillary wedge pressure; Pt = patient; RAP = right atrial pressure.

disease; 4 had two-vessel disease; and 1 had two-vessel and left main disease. In nine patients, the infarct-related artery was occluded. All 17 patients had a localized aneurysm or an akinetic area. In two patients, a small jet of contrast material was seen to enter the pericardial sac during angiography. One of these patients had a sealed LVFWR confirmed during open heart surgery 42 days later.

Follow-up. There were no deaths among the 15 survivors during a follow-up period of 52.5 ± 35.2 months (Table 1), and serial echocardiograms disclosed progressive disappearance of pericardial effusion, with no evidence of pseudoaneurysm.

Discussion

Survival of LVFWR without surgical closure. The main finding of this study is the observation of long-term survival in some patients with an AMI who recovered from cardiac tamponade due to strongly suspected LVFWR without surgical repair. Similar results have not been reported, except for one isolated case (25), and survival after LVFWR has only been documented in patients undergoing operation (5,10–14). The true survival rate from these interventions is unknown because only successful results tend to be reported, and most patients do not undergo operation. In a review in 1989, Bolooki (26) found only 32 patients with surgical repair. Nevertheless, it has been established that surgical repair is the only therapeutic choice for acute or subacute LVFWR (10,13–19,27). Although this seems mandatory in the absence of satisfactory recovery, there is no definite evidence that it needs to be implemented in all patients with prompt hemodynamic improvement. In fact, LVFWR is a notorious a complication in the elderly at high surgical risk (10,18,19). In contrast, development of a left ventricular pseudoaneurysm is exceedingly rare (20–22,28). Also, because false ventricular aneurysms are often linked to silent rupture (21,22,28,29), it is possible that some ruptures may seal and not evolve to false aneurysms. We therefore speculate that this is what happened to our patients, and it is conceivable that some self-limited cases of LVFWR may be overlooked because their only manifestation might be transient hypotension or asymptomatic hemopericardium. Furthermore, pathologic studies have suggested that some patients may present with small leaks (30,31) that might close spontaneously by epicardial fibrin deposits. Indeed, blood clots at the endocardial (13,14,31,32) as well as the epicardial site of rupture (14) have often been identified, suggesting protection from further rupture. Accordingly, echocardiographic evidence of a fibrinous cap attached to the rupture site, as in 10 of our patients, has been reported (33).

Role of prolonged rest and blood pressure control. It is conceivable that in some of our patients, lowering of blood pressure and reduction in myocardial contractility with beta-blockers, which appear to reduce incidence of LVFWR (7), contributed to prevent rerupture. At the same time, it is also reasonable to ascribe a protective effect against a second rupture to prolonged rest because physical strain has been consistently proposed as a potential trigger of LVFWR (2–4,32). However, a definitive role of either prolonged bed rest or beta-blocker therapy, or both, in the successful management of our patients, cannot be established in the absence of an untreated control group. Only two patients died of a second rupture (10.5%). In both patients, arterial hypertension reappeared before rerupture, although the reasons for this reappearance remain unclear. Thus, it is possible that, as reported in other series (3,4,32,34,35), uncontrolled hypertension might have been the precipitating cause of the fatal event. It should be stressed that surgical intervention as backup treatment

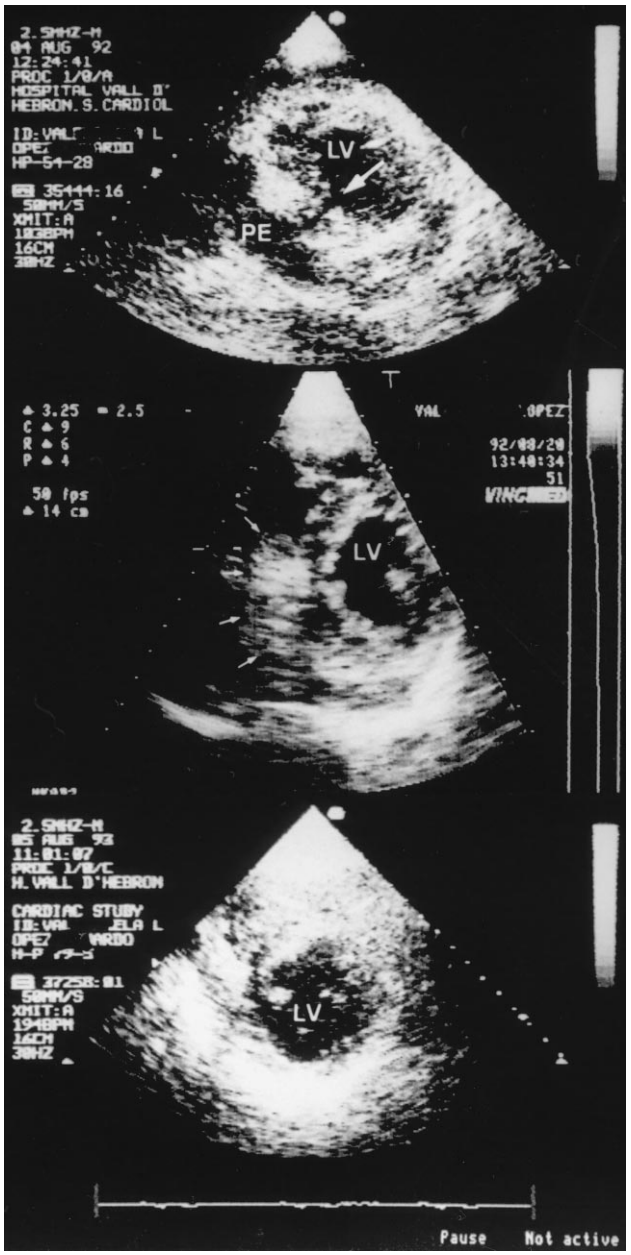

Figure 1. Sequential echocardiographic images of LVFWR in a 56-year old man (Patient 11) with a lateral myocardial infarction. **Top,** Shortly after clinical recognition of the LVFWR; **arrow** indicates the rupture site, and significant pericardial effusion (PE) can be seen. **Middle,** Sixteen days later, there is an extensive fibrinous cap attached to the ruptured site (**arrows**). **Bottom,** Twelve months later, the fibrin deposits and pericardial effusion are no longer apparent. LV = left ventricle.

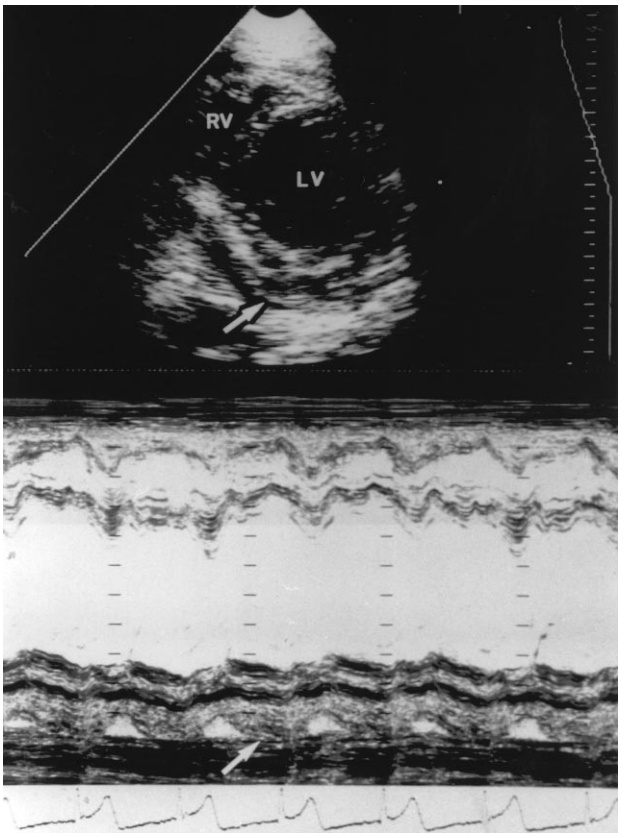

Figure 2. Echocardiographic evidence of a pericardial effusion in a 63-year old man (Patient 5) with an inferolateral infarction, with an echo-dense mass in the pericardial sac **(arrow).** In the M-mode image **(bottom),** the mass **(arrow)** appears to be attached to the epicardium of the inferior wall. LV = left ventricle; RV = right ventricle.

was available during the first few days after LVFWR, and in very emergent conditions, the surgical team did operate on some of the patients who had a poor or absent initial recovery.

Anterior versus inferior or lateral AMI. Our medically treated patients (group C) had a lower incidence of anterior AMI and a higher incidence of lateral AMI than those who died early (group B). This may indicate that anterior infarctions are perhaps less protected with pericardial blood because blood would tend to pool and clot in the posterior wall in recumbent patients. Likewise, patients who develop a false left ventricular aneurysm also tend to be those with a posterior or lateral infarction (22,23).

Follow-up: pseudoaneurysm formation. There was no evidence of rerupture during an average follow-up period of 52 months, nor was there evidence of pseudoaneurysm. Although there is no prospective documentation that similar cases have evolved to pseudoaneurysm, it is known that pseudoaneurysm may develop within a few weeks or months after AMI (20,22,23). Thus, we suggest that patients who recover from an episode of LVFWR with hemopericardium are unlikely to develop a pseudoaneurysm.

Study limitations. The possibility that cardiac tamponade in some patients could have been related to other causes cannot be entirely ruled out. Accordingly, there have been isolated reports of bloody pericardial effusion during an AMI (36,37) that may be enhanced by anticoagulant therapy (37), but this is an exceedingly rare complication (38,39). We have seen very few cases of hemorrhagic effusion in AMI other than those due to LVFWR and mainly as a consequence of a right ventricular perforation by a pacing catheter. In addition, the presence of an effusion >5 mm, hypotension and increased

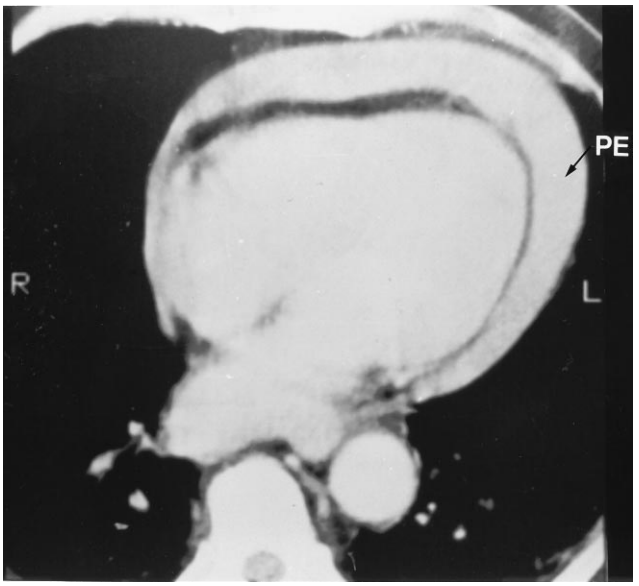

Figure 3. Axial computed tomographic image of a moderate to severe effusion (PE) in a 65-year old man (Patient 3) who presented with EMD in the course of a lateral myocardial infarction and who underwent life-saving pericardiocentesis (50 ml). L = left; R = right.

filling pressures (>10 mm Hg) had a specificity of 99.8% in diagnosing LVFWR, confirmed at operation, in 33 patients with a first AMI from a recently reported multicenter study (18).

Clinical implications. Our results should be interpreted cautiously in view of the small number of study patients. However, if confirmed by larger series, medical management might emerge as an alternative to operation for some patients who satisfactorily recover from an episode of cardiac tamponade secondary to LVFWR. Furthermore, our findings should encourage resuscitation maneuvers in patients with this condition, often considered irreversible. Ten of our patients with EMD were successfully resuscitated, including cardiac massage in 5, and all were long-term survivors. According to our results, medical management might be of particular value in patients with a lateral or an inferoposterior AMI or those at very high surgical risk, such as those with a large infarct area or those >75 years old. In contrast, surgical intervention should be considered for those patients whose arterial hypertension is difficult to control or those in whom tamponade reccurs after pericardiocentesis. Moreover, in view of more encouraging recent surgical results (40), comparative studies are necessary to better define the best strategy for the different subsets. However, at present, it seems mandatory that all such patients remain at or be transferred to an institution with surgical backup.

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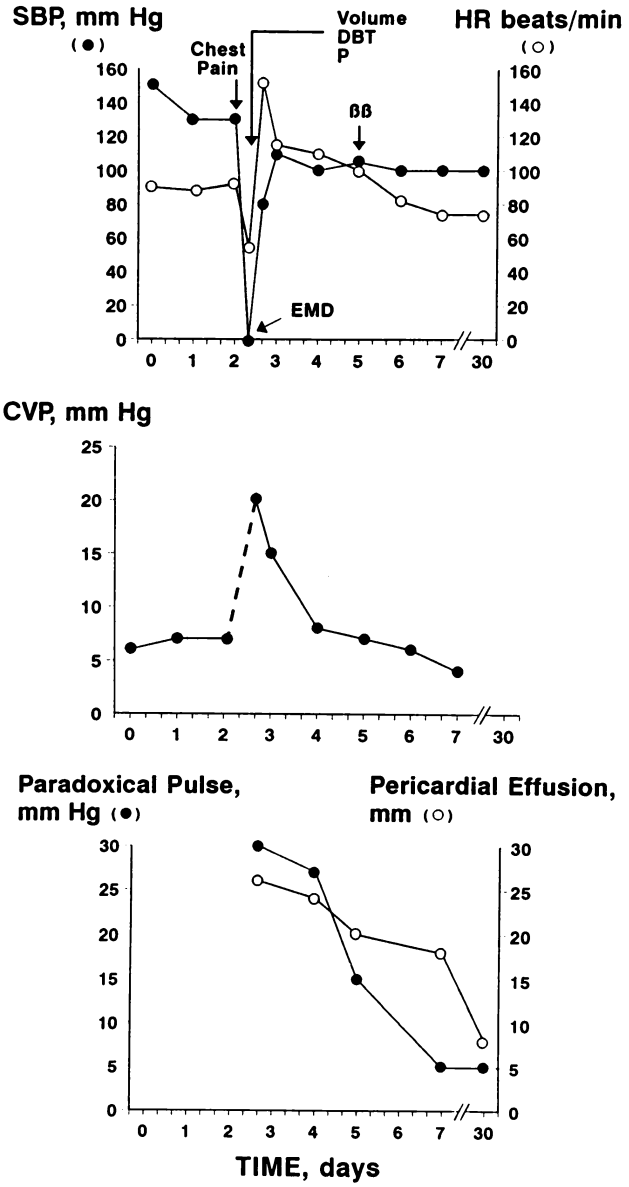

Figure 4. Sequential changes in systolic blod pressure (SBP), heart rate (HR), central venous pressure (CVP), paradoxic pulse and pericardial effusion in a 55-year old man (Patient 12) with an anterolateral AMI during cardiac tamponade caused by LVFWR that led to EMD and was preceded by chest pain. Hemodynamic recovery supervened after an intravenous infusion of dobutamine (DBT) and a colloid solution (Volume) and withdrawal of 10 ml of bloody fluid through pericardiocentesis (P). In this patient, propranolol (BB) was started 2 days after the event. Note that pericardial effusion on day 7 was still striking, without causing hemodynamic compromise, and was 8 mm at discharge at 30 days.

References

1. Mundth E. Rupture of the heart complicating myocardial infarction. Circulation 1972;46:427–9.
2. Bates R, Beutler S, Resnekov L, Anagnostopoulos C. Cardiac rupture—challenge in diagnosis and management. Am J Cardiol 1977;40:429–37.
3. Figueras J, Curos A, Cortadellas J, Sans M, Soler-Soler J. Relevance of electrocardiographic findings, heart failure, and infarct site in assessing risk and timing of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol 1995;76:543–7.

4. Friedman HS, Kuhn LA, Katz AM. Clinical and electrocardiographic features of cardiac rupture following acute myocardial infarction. Am J Med 1971;50:709–20.
5. Cobbs BW Jr, Hatcher CR Jr, Robinson PM. Cardiac rupture. Three operations with two long-term survivors. JAMA 1973;223:532–5.
6. Feneley MP, Chang VP, O'Rourke MF. Myocardial rupture after acute myocardial infarction: ten-year review. Br Heart J 1983;49:550–6.
7. ISIS-1 (First International Study of Infarct Survival) Collaborative Group. Mechanisms for the early mortality reduction produced by beta-blockade started early in acute myocardial infarction: ISIS-1. Lancet 1988;1:921–3.
8. Figueras J, Curós A, Cortadellas J, Soler-Soler J. Reliability of electromechanical dissociation in the diagnosis of left ventricular free wall rupture in patients with acute myocardial infarction. Am Heart J 1996;131:861–4.
9. Mann JM, Roberts WC. Rupture of the left ventricular free wall during acute myocardial infarction: analysis of 138 necropsy patients and comparison with 50 necropsy patients with acute myocardial infarctions without rupture. Am J Cardiol 1988;62:847–59.
10. O'Rourke MF. Subacute heart rupture following myocardial infarction: clinical features of a correctable condition. Lancet 1973;2:124–6.
11. Calick A, Kerth W, Barbour D, Cohn K. Successful surgical therapy of ruptured myocardium. Chest 1974;66:188–93.
12. Windsor HM, Chang VP, Shanahan MX. Postinfarction cardiac rupture. J Thorac Cardiovasc Surg 1982;84:755–61.
13. Bashour T, Kabbani SS, Ellertson DG, Crew J, Hanna ES. Surgical salvage of heart rupture: report of two cases and review of the literature. Ann Thorac Surg 1983;36:209–13.
14. Balakumaran K, Verbaan CJ, Essed CE, et al. Ventricular free wall rupture: sudden, subacute, slow, sealed and stabilized varieties. Eur Heart J 1984;5:282–8.
15. Van Tassel RA, Edwards JE. Rupture of heart complicating myocardial infarction. Analysis of 40 cases including nine examples of left ventricular false aneurysm. Chest 1972;61:104–16.
16. Coma-Canella I, Lopez-Sendon J, Nuñez Gonzalez L, Ferrufino O. Subacute left ventricular free wall rupture following acute myocardial infarction: bedside hemodynamics, differential diagnosis, and treatment. Am Heart J 1983;106:278–84.
17. Dellborg M, Held P, Swedberg K, Vedin A. Rupture of the myocardium. Occurrence and risk factors. Br Heart J 1985;54:11–6.
18. Lopez-Sendon J, Gonzalez A, Lopez de Sa E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: Sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol 1992;19:1145–53.
19. Shapira I, Isakov A, Burke M, Almog C. Cardiac rupture in patients with acute myocardial infarction. Chest 1987;92:219–23.
20. Chesler E, Korns M, Semba T, Edwards JE. False aneurysms of the left ventricle following myocardial infarction. Am J Cardiol 1969;23:76–82.
21. Vlodaver Z, Cole JL, Edwards JE. True and false left ventricular aneurysms. Circulation 1975;51:567–72.
22. Gueron M, Wanderman KL, Hirsch M, Borman J. Pseudoaneurysm of the left ventricle after myocardial infarction. A curable form of myocardial rupture. J Thorac Cardiovasc Surg 1975;69:736–42.
23. Rittenhouse EA, Sauvage LR, Mansfield PB, Smith JC, Davis CC, Hall DG. False aneurysm of the left ventricle. Report of four cases and review of surgical management. Ann Surg 1979;189:409–15.
24. Killip T III, Kimball JT. Treatment of myocardial infarction in a coronary care unit: a two years experience with 250 patients. Am J Cardiol 1967;20:457–64.
25. Dvorak K, Cerny J. Long-term survival of subacute cardiac rupture with tamponade in acute myocardial infarction without surgical intervention (the role of pericardiocentesis). Cor Vasa 1979;21:233–41.
26. Bolooki H. Emergency cardiac procedures in patients in cardiogenic shock due to complications of coronary artery disease. Circulation 1989;79 Suppl I:I-137–48.
27. Pifarre R, Sullivan HJ, Grieco J, et al. Management of left ventricular rupture complicating myocardial infarction. J Thorac Cardiovasc Surg 1983;86:441–3.
28. Ersek RA, Chesler E, Korns ME, Edwards JE. Spontaneous rupture of a false left ventricular aneurysm following myocardial infarction. Am Heart J 1969;77:677–80.
29. Cone RB, Hawley RL. Pseudoaneurysm of the heart following infarction. Arch Pathol 1964;77:166–71.
30. Freeman WJ. The histologic patterns of ruptured myocardial infarcts. Arch Pathol 1958;65:646–53.
31. Lautsch FV, Lanks KW. Pathogenesis of cardiac rupture. Arch Pathol 1967;84:264–71.
32. Wessler S, Zoll PM, Schlesinger MJ. The pathogenesis of spontaneous cardiac rupture. Circulation 1952;6:334–51.
33. Knopf WD, Talley JD, Murphy DA. An echo-dense mass in the pericardial space as a sign of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol 1987;59:1202.
34. Edmonson HA, Hoxie HJ. Hypertension and cardiac rupture. Am Heart J 1942;24:719–33.
35. Christensen D, Fond M, Reading J, Castle H. Effect of hypertension on myocardial rupture after acute myocardial infarction. Chest 1977;72:618–22.
36. Rose OA, Ott RH Jr, Maier HC. Hemopericardium with tamponade during anticoagulant therapy of myocardial infarction: report of a case with recovery following pericardiotomy. JAMA 1953;152:1221–3.
37. Blau N, Shen BA, Pittman DE, Joyner CE. Massive hemopericardium in a patient with post-myocardial infarction syndrome. Chest 1977;549–56.
38. Friedberg CK. Diseases of the Heart. Philadelphia: WB Saunders, 1966:914.
39. Galve E, Garcia del Castillo H, Evangelista A, Batlle J, Permanyer-Miralda C, Soler-Soler J. Pericardial effusion in the course of myocardial infarction: incidence, natural history, and clinical relevance. Circulation 1986;73:294–9.
40. Padró JM, Mesa JM, Silvestre J, et al. Subacute cardiac rupture: repair with a sutureless technique. Ann Thorac Surg 1993;55:20–4.